A CASE OF INTERNAL HEMORRHAGIC PACHYMENINGITIS IN A CHILD OF NINE YEARS, WITH CHANGES IN THE NERVE CELLS.[1]

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The existence of internal hemorrhagic pachymeningitis in childhood has received comparatively little attention in the text-books and general medical literature. Herter's[2] excellent article published in 1898, with the report of two cases, is an exception to the rule. Even when mention is made of the subject it is not usually in the report of cases or of personal experience, but merely in allusion to the writings of others.

Internal hemorrhagic pachymeningitis, even in the adult, is said by Gowers to be "very rare," and is of interest "chiefly as a pathological curiosity enigma." Gowers[3] further states that its rarity outside of asylums may be judged from the fact that during the first forty years in which the London Pathological Society received the curiosities of metropolitan necropsy not a single specimen was brought before the society from any London hospital.

Osler[4] says that during eight years in the post-mortem room of the General Hospital of Montreal no case of internal hemorrhagic pachymeningitis occurred, and the first specimen he saw after having been for fourteen years interested in morbid anatomy was in Virchow's laboratory; but he saw numerous examples later at the Philadelphia Hospital, usually in bodies from the insane department.

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[1] From the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania (Phœbe A. Hearst Foundation).

Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

[2] Herter, The American Journal of the Medical Sciences, 1898.

[3] Gowers, "A Manual of Diseases of the Nervous System." Second English edition, p. 325.

[4] Osler, Journal of Nervous and Mental Disease, 1888, p. 608.

While the condition is not one of great frequency, it does occur often enough to be seen by many neuropathologists, and the mere report of a case in the adult would hardly be justifiable. The occurrence of this pathological condition in children is worthy of mention. According to Gowers, in early life it is relatively as well as absolutely infrequent, and occurs chiefly in infancy, as frequently in the first year of life as between one and twenty years of age.

Dercum,[5] in an article in his text-book, refers to the papers by Doehle and Northrup as evidence of the occurrence of internal hemorrhagic pachymeningitis in childhood. He speaks also of a variety of chronic external pachymeningitis occurring in childhood, and described by Lannelongue and Alexis Thompson, and he mentions inherited syphilis as a cause of pachymeningitis in childhood.

Mills[6] refers to the existence of the condition in children, the subjects of hemorrhagic rachitis, and in the discussion of a paper by Osler on internal hemorrhagic pachymeningitis reported a case in a child of about three years of age.

Northrup[7] has reported four cases occurring in children, the eldest child being four years and seven months old. He refers to a case in childhood recorded by Emmett Holt, and to one by S. J. McNutt. He says he has seen only four cases, all in children under two years of age, in an experience founded upon fifteen hundred necropsies.

Hoyt[8] says that the few cases reported as occurring in early life were due to traumatism or some grave general disease.

Bullard[9] speaks of *traumatic* cases occurring in well-grown children.

Holt[10] says that chronic internal pachymeningitis is not very rare, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases. He says he has seen five cases. He also makes the statement that pachymeningitis plays a much more important rôle in the production of meningeal hemorrhage in children than has generally been accorded to it.

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[5] Dercum, "A Textbook on Nervous Diseases," pp. 350, 352.

[6] Mills, "The Nervous System and its Diseases," p. 265. Journal of Nervous and Mental Disease, 1888, p. 611.

[7] Northrup, Proceedings of the N. Y. Path. Soc., 1890, pp. 59 and 67.

[8] Hoyt, Medical Record, April 30, 1892, p. 485.

[9] Bullard, Boston Med. and Surg. Journal, Nov. 7, 1895, p. 461.

[10] Holt, "Diseases of Infancy and Childhood."

Oppenheim[11] describes a form of chronic meningitis in young children, confined almost entirely to the posterior cranial fossa and supposed to be of syphilitic origin. The meninges are united with the cerebellum and medulla oblongata. This condition mentioned by Oppenheim seems to be different from that observed by us in the case to be reported in this paper.

According to Henoch,[12] pachymeningitis in moderate intensity and extent occurs not so very rarely in children, but is not as common as in adults. Our case seems to be extraordinary, not only on account of the advanced age of the child, but also on account of the extent of the process.

The oldest child with internal hemorrhagic pachymeningitis to which Herter was able to refer was six years of age (Doehle). The age of the patient studied by us was nine years.

Doehle[13] believes, from the necropsies made in the pathological institute of Kiel, that chronic internal pachymeningitis is not so rare in children as is usually believed. In 395 autopsies on children, 269 being in children one year old and under, chronic pachymeningitis was found 48 times; and in 38 of these cases the children were not over one year old; in the other ten they were not over eight years old. No pachymeningitis was observed in children nine or ten years of age, and no mention is made of ages more advanced than these. Doehle even believes that pachymeningitis is of more common occurrence in childhood than in later life, but he states that the existence of chronic pachymeningitis in childhood has received little attention.

This brief résumé of the literature presents a variety of opinions held in regard to the occurrence of internal hemorrhagic pachymeningitis in childhood. No mention is made of the condition in certain books devoted to the diseases of children.

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[11] Oppenheim, "Lehrbuch der Nervenkrankheiten," first edition, p. 490.

[12] Henoch, "Vorlesungen über Kinderkrankheiten," 1892, p. 285.

[13] Doehle, "Verhandlungen des X internationalen medicinischen Congresses," Abtheilung XVII, Gerichtliche Medicin, p. 40.

The case that we report is from the Pennsylvania Training School for Feeble-Minded Children. We are indebted to Dr. M. W. Barr and Dr. Llewellyn for the clinical notes.

M. F., born December 12, 1889, was the child of healthy parents. The father was a teacher and very erratic. The parents, though not related before marriage, bore a striking resemblance to one another. The father was thirty-six, the mother thirty-three, when M. was born. The parents and grandparents were not addicted to the use of alcoholic drinks. A history of insanity or feeble-mindedness was obtained in the family of a great-grandfather. There was no tendency to blindness or deafness in the child's family on either side, excepting that the father was somewhat deaf in the left ear as the result of a severe attack of scarlet fever when seventeen years old. Two sisters of M. were said to be of sound health in body and mind. She had no brothers. A dead sister, three years M.'s senior, resembled M. in constitution; and died in a home for feeble-minded children in Iowa, of "tuberculous meningitis," January 31, 1894, at the age of eight years. She was the second child in the family and resembled M. to such an extent that the parents had only one photograph taken for the two children, inasmuch as the one represented both.

M. was born without instruments, but version was performed. She was not asphyxiated and had no convulsions following birth. She was the third child in the family. She probably had adenoid vegetations, as she experienced difficulty in nursing and in breathing through her nose. She cried for thirty-six hours after birth and is said to have "ruptured her navel and one side, and to have enlarged her head somewhat." M. was not subject to severe illness. Her sight was said to be good, but her hearing defective; this defective hearing may really have been a want of attention. She could not speak. She was right-handed and had no anomaly of gait. She began to walk before she was two years of age and had no motor paralysis. She had some of the infectious diseases (measles, whooping-cough), but never had convulsions. Her bowels were "weak;" this statement probably means that she was subject to diarrhea. The feet and hands were always cold and blistered easily. She masticated her food properly. She was said to be lacking in sensation, and would handle hot coals and hot food without uttering a sound of discomfort. She was exceedingly active and noisy, as so many feeble-minded children are, and was destructive, heedless of danger and careless. She wet the bed as well as her day clothing, did not understand what was said to her, and could not read. The above history was obtained from the child's father, December 30, 1895.

At the time of admission to the school, October 17, 1896, she was recorded as an idiot with a large head, the circumference of which was 21 inches. Her sensation could not be accurately tested on account of her mental condition. Acuity of vision was never tested, for the same reason, but her sight was apparently good. No peculiarities of motion were noticed. The movements were not inco-ordinate and tremor and convulsions did not exist. Tendon phenomena were not obtained. No trophic disorders were observed. The child could not talk; she was unclean in her habits and noisy. Pulse was normal. Incontinence of urine existed. The number of red blood corpuscles to a cubic millimeter was 5,800,000. Hemoglobin was 74 per cent. No rigidity was detected.

After having been in the school one year she was reported as probably more troublesome and harder to manage than when admitted; although her parents seemed to think she was improved when they visited her in September, 1897.

For about a year previous to her death, M. showed very gradual emaciation, although careful physical examination revealed no special cause for it. The gait was observed to be more shambling than previously, and this was attributed to physical weakness. In February, 1899, she was reported as thin and poorly nourished.

On February 8, 1899, emaciation being extreme she was sent to the hospital, was fed upon very nourishing diet, and was kept in bed most of the time. In three weeks she had gained a little in strength and ran about the ward as usual. At this time diarrhea was troublesome for three days and was checked by simple remedies.

On March 6, 1899, at 6.20 A. M., word came from the hospital that the child seemed to be dying. Examination showed very weak and irregular heart sounds, 110 per minute; respiration 28; temperature 100 degrees F. She refused to swallow. Occasionally she moved her hands and legs. She remained in this condition, growing weaker until death resulted on the same day at 3.30 P. M.

The necropsy was performed May 9.

The body was that a much emaciated child. The head was long, narrow and appeared rachitic. The bridge of the nose was flattened and suggested congenital syphilis. The lower front teeth were nicked like those known as Hutchinson's teeth.

The sternum was very prominent, and presented the so-called pigeon-breast. Other bones were not enlarged. The dura was partly adherent to the calvarium and the latter was removed with considerable difficulty. The membranes over the convexity of the brain presented a peculiar boggy appearance and were bluish in color. When the dura was incised on the left side about two or three ounces of dark fluid blood escaped. Two layers of new membrane could be stripped from the inner side of the dura. The layer next to the dura was exceedingly vascular, thick, edematous and of a dark red color; that more internally situated did not appear to be vascular macroscopically, but the microscope showed the presence of vessels. The innermost layer of membrane, especially on the left side, was loosely attached to the more superficial layer and the latter could be torn from the dura without difficulty, although it was distinctly adherent. The pachymeningitis extended well toward the base of the cranium on each side. The new membranes were not adherent to the pia except at the superior longitudinal fissure. The pia was very edematous and the brain was quite soft.

The brain was small and the cerebellum was very imperfectly covered by the occipital lobes. The fissures were deep and the character of fissuration was that of low-type cerebra. The brain was not weighed at the time of removal, but the left hemisphere with the left cerebral peduncle and half of the pons, but without the cerebellum, weighed 290 grams after the tissue had been kept some time in formaline.

The liver extended upward to the fourth rib and downward three fingers' breadth below the last rib, chiefly on account of the malformation of the thorax. No scars suggestive of syphilis were found in the liver. The other viscera were examined, but nothing worthy of special note was detected.

The central nervous system was studied microscopically.

Right paracentral lobule: thionin stain: All the nerve cells were found to be much swollen (Fig. 1) and they had undergone intense chromatolysis. The nucleus was displaced to the periphery of the cell. The larger cells presented a swollen, rounded body with a pointed end, and this pointed extremity was usually the portion of the cell nearest the free surface of the cortex. In these cells the chromophilic elements were found only toward this pointed extremity. While in most of the cells the nucleus was in the pointed end, in some it occupied the other extremity of the cell body. Some of the cells were entirely deprived of chromophilic elements.

A structure, of a reticular character, stained purple with the thionin, was found in the portion of the cell deprived of chromophilic elements (Fig. 11). This finding would correspond with the views of those who hold that a reticulum exists normally in the nerve cell, and that the chromophilic elements are merely incrustations upon the intracellular network. The cortical cells presented a striking resemblance to miniature balloons, the point at which the chromophilic elements were preserved corresponding to the basket of the balloon. The smaller cells presented the same appearance, but the condition was especially striking in the large giant cells of the paracentral lobule. The chromophilic elements preserved in the pointed extremity did not usually present the rod-shape normal to the giant cell, but were distinctly granular. The nucleus in many

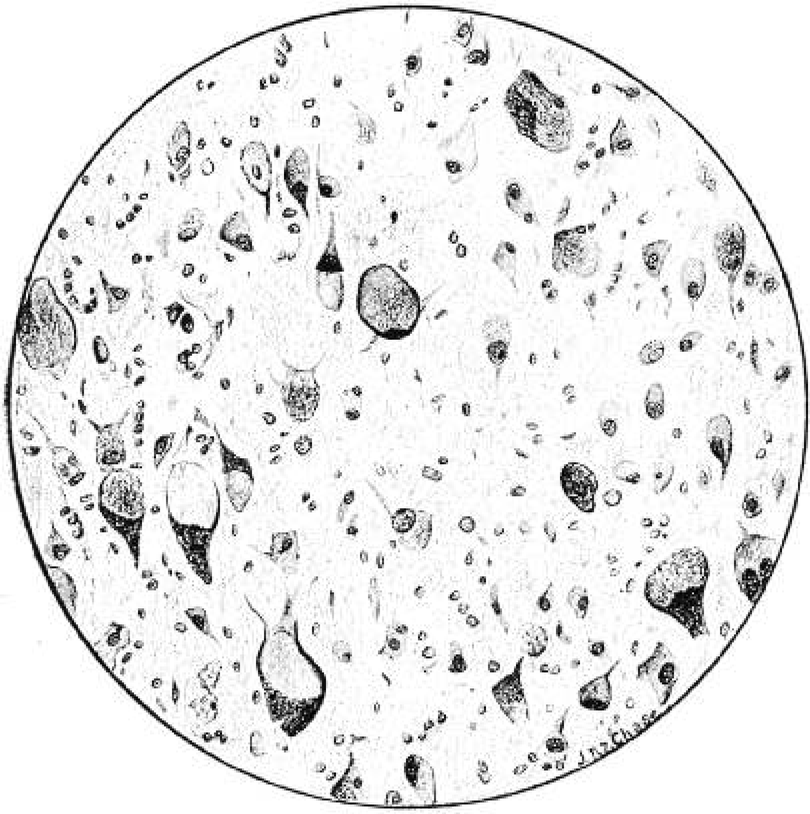

Fig. I.—Section from the paracentral lobule. The nerve cells are swollen, and in a condition of chromatolysis.

of the nerve cells stained somewhat deeply and the nucleolus stained intensely. In the apical process of some of the cells normal chromophilic elements were found.

Right paracentral lobule, Weigert's hematoxylin method: The appearance presented by this stain was almost more striking than that by the thionin preparations. The nerve cells stained a yellowish brown. In many of the tumefied giant cells a considerable accumulation of granular matter was found that stained purplish-black with the hematoxylin, and this material was usually nearer the pointed extremity of the cell where the chromophilic elements were found by the thionin stain. In some cells this granular matter occupied the entire cell body.

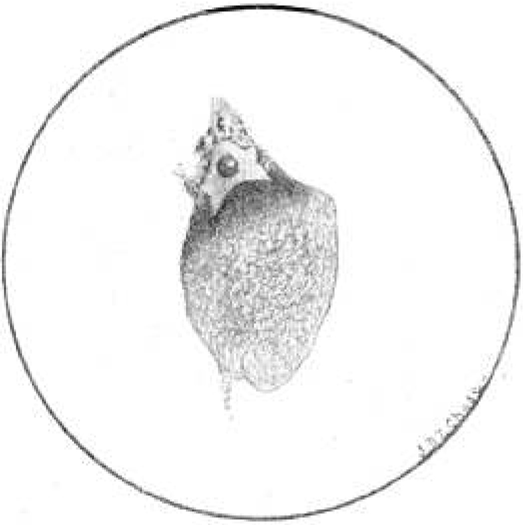

Fig. II.—One of the giant cells from the paracentral lobule under higher magnification, showing chromatolysis and a reticulum.

The nucleus could be distinguished with this stain and the nucleolus stained a deep brown or even black.

The small vessels of the cortex were widely distended with red blood corpuscles that stained intensely with the hematoxylin. The small capillaries were so numerous in the gray matter that a proliferation of these vessels in the cortex had probably occurred (Fig. III). Both gray and white matter contained many red blood corpuscles free within the tissue. Numerous red blood corpuscles were found in the perivascular spaces of certain of the vessels, and considerable collections of these red blood corpuscles were seen free in the tissues. The red blood corpuscles free within the nervous tissues were apparently normal, and were indicative of recent infiltration. Altered blood pigment was not found within the tissue of the paracentral lobule.

Nerve cells in sections taken from the frontal lobe and posterior portion of the parietal lobe, from the cerebellum and through the bulbar nuclei exhibited similar changes to those in the paracentral lobule.

Spinal cord, cervical region: The spinal cord was hardened in Müller's fluid, and therefore the thionin stain could not be employed. With the Weigert hematoxylin stain, the white matter of the cord appeared to be normal. The cells of the anterior horns stained as intensely by this method as those of any other part of the central nervous system, and even by this method were seen to be greatly tumefied and often without dendritic processes. This intense coloration of the anterior

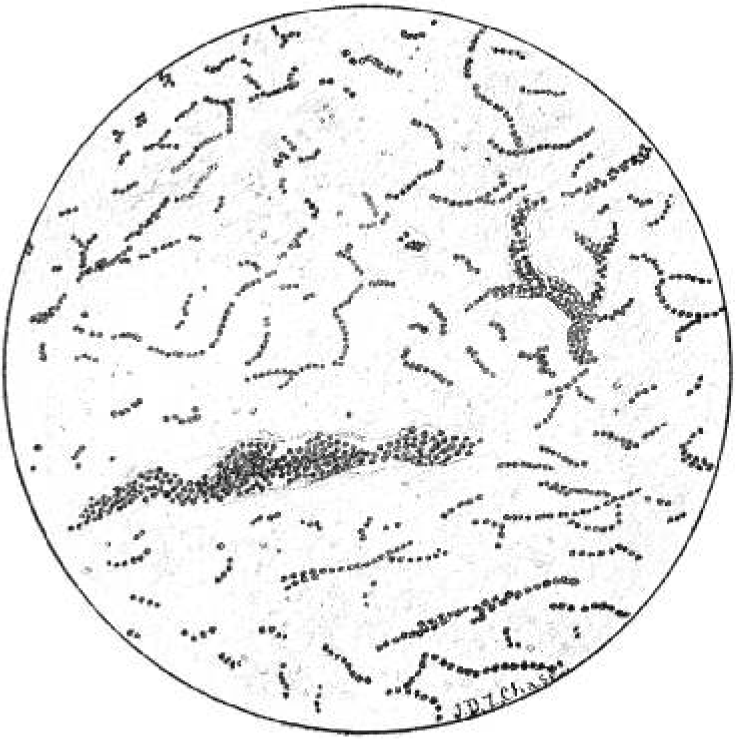

Fig. III.—Proliferation of capillaries in the cerebral cortex.

horn cells was especially striking, inasmuch as sections of the spinal cord stained by the Marchi method did not exhibit the black granules so often found in the spinal cords of the aged. The cells of the anterior horns by the Marchi method were pale yellow and entirely free from black granules. This was not due to insufficient penetration of the osmic acid, as the tissue had assumed the color always seen in normal tissue properly stained by this method.

The anterior and posterior roots by the Weigert method were aparently normal, but small black granules were seen within the intramedullary portion of the posterior and anterior roots when stained by the Marchi method. As these dots are at times found in the roots in normal tissue, we merely record the finding without asserting that the roots were degenerated.

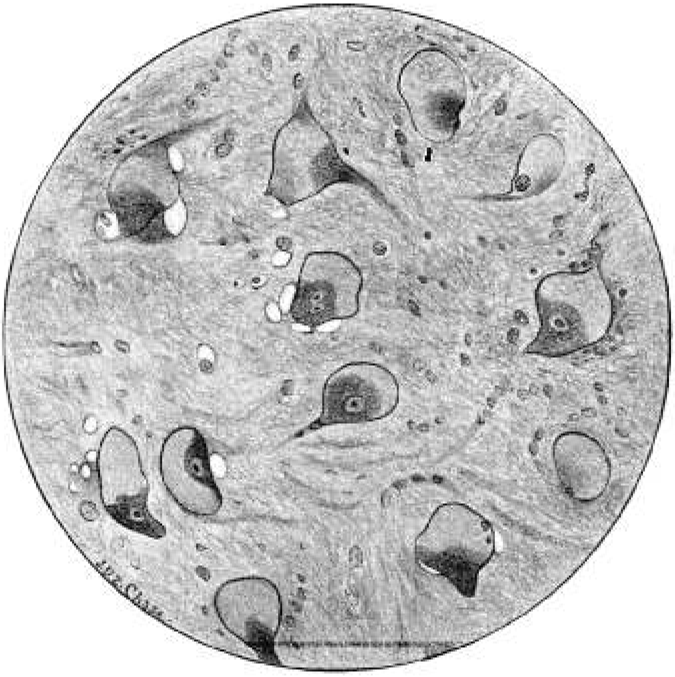

Fig. IV.—Nerve cells in the anterior horn of the spinal cord.

Sections from the cervical region stained with ammonium carmine showed exactly the same changes in the anterior horn cells as above described as occurring in the paracentral lobule and cortex generally. The cells were almost globular (Fig. IV), with a pointed extremity, and the nucleus was found in this pointed end. About the nucleus was a mass of matter that took the carmine stain intensely. The rest of the cell was a pale yellow and had no affinity for the carmine.

The small vessels of the spinal cord were intensely congested.

Lumbar region: The description of the cervical region applies equally well to the lumbar region. The cells of the columns of Clarke were as markedly affected as those of the anterior cornua, and yet no degeneration of the direct cerebellar tracts was apparent either in the Weigert or the carmine preparations.

Distinct signs of inflammation were not present in the cerebral dura. The blood vessels were engorged and in some places large accumulations of red blood corpuscles were found free within the dura, but there was no distinct round cell infiltration. The nuclei of the cells in the dura stained well, but were not as elongated as is usual in fibrous connective tissue. At one place in a section where the dura made a fold upon itself the endothelial lining was very distinct and the portion within the fold most distant from the false membrane was apparently normal. Nearer the false membrane the cell nuclei of the endothelium were more globular.

In the layer of false membrane immediately adjoining the dura were many blood vessels having walls so delicate that they were recognizable only by the presence of endothelial cells. These vessels were greatly distended and numerous red blood corpuscles were found free within the tissue. The foundation substance of this membrane was amorphous or somewhat fibrillar, and it contained many nuclei; some of which were spindle-shaped or round, but the majority were oval. They were doubtless the nuclei of fibroblasts in different stages of formation.

In the false membrane between the portion next to the dura and that most remote, the tissue seemed to be in an earlier stage of formation; the nuclei were more distinctly round or oval and the long spindle-shaped nuclei were absent.

The portion of false membrane most remote from the dura showed a further advanced formation than the portion just referred to. The tissue was more fibrillar, denser; and the nuclei, while on the whole not as elongated as those in fully formed connective tissue, were nevertheless more fusiform than those in the central layer of the false membrane. The blood vessels throughout the new-formed membrane were numerous, and it can hardly be said that they were more numerous in one layer of the membrane than in another. Large masses of blood pigment were found within the false membrane.

The looser and more amorphous tissue of the middle layer and the character of the nuclei contained within it indicate that this portion of the membrane was of later formation, and it seems possible in the light of our experiments to be mentioned further on, that the membrane may have formed after a subdural hemorrhage had occurred, and that the peripheral parts of the clot became organized before the central.

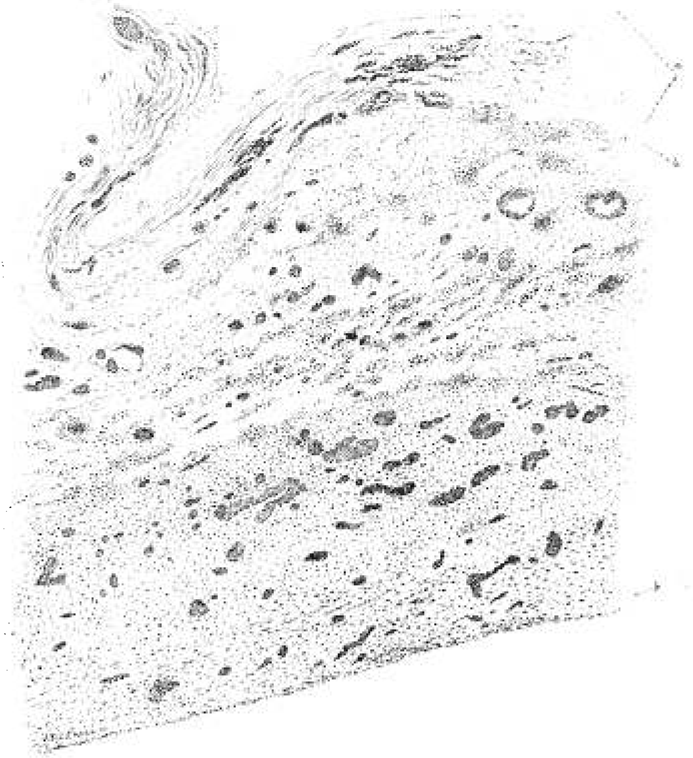

Fig. V.—The dura (*a*) is shown in the uppermost part of the drawing. Immediately next to the dura is a tissue of somewhat loose structure, and beneath this is a dense tissue rich in vessels.

At the necropsy, two distinct membranes could be torn from the inner surface of the dura, and the loose tissue referred to was probably the connection between the two layers.

Groups of round cells were not found within the false membrane.

The changes in the nerve cells throughout the central nervous system, both brain and cord, were of much interest. A condition of intense chromatolysis existed and the cells were tumefied. In many cells only a few chromophilic elements remained at one portion of the cell body, and the dendritic processes had disappeared or were very indistinct. We noticed such changes in the cells of the cerebral and cerebellar cortex, in the bulbar nuclei, and in the cells of the spinal cord.

Some of the Purkinje cells of the cerebellum stained by mistake with ferricyanide of potassium and acid alcohol presented distinct small dark granules, filling the entire cell body and extending in some cases from the cell body into the surrounding tissue. The exact significance of these granules is unknown. They may possibly be the metaplasm granules of Van Gieson, or they may indicate an early stage of iron infiltration of the cell body such as Weber[14] has recently described.

Red blood corpuscles stained by Weigert's hematoxylin were dark in color and the presence of the dark granules in the nerve cells of our case by this stain suggested the possibility of an infiltration of coloring matter from the blood. Weber found cerebral ganglion cells in which the cell body, except the nucleus, stained such a deep black by alum hematoxylin that they resembled the cells stained by the silver method. When the color was less intense the cells appeared to be filled with blue granules. These cells when placed in a 5-per-cent. solution of ferrocyanide of potassium and acid alcohol gave the Berlin blue color. With the Weigert hematoxylin stain the infiltrated cells were deep black, while the other cells were brownish-yellow. The siderophilous cells were always in the vicinity of diseased and bleeding vessels. Weber believed that the granules he described contained an albuminate of iron. He was unable to find an exactly analogous case in the literature and he thought that the early age of the child in his case might have rendered the nerve cells more susceptible to this infiltration.

The early age of the child in our case, the proliferation and engorgement of cortical capillaries, the free red blood corpuscles within the cerebral tissues, made the deep pigmentation of the nerve cells by the Weigert hematoxylin method seem possibly the result of infiltration by blood coloring matter, The characteristic iron reaction was not obtained even when the proper potassium salt was used. We cannot entirely dismiss from our minds the possibility that this intense coloration of the nerve cells by the Weigert method was due to blood coloring matter, although we regard it as an unsettled question.

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[14] Weber, Monatsschrift für Psychiatrie und Neurologie, Vol. 3, No 6, p. 507.

Doehle is inclined to believe that intracranial hemorrhage at birth is the cause of the pachymeningitis, and that the brain suffers at the time of the formation of the hemorrhage, although he had few findings to offer in support of the latter view. It seemed to him that cortical changes might be the cause of the deficient bodily development of these children, but the cortical changes he had in mind were those produced by the trauma that causes the hemorrhage and not the changes resulting from the presence of the membranes with the fluid contained within them—at least his words permit this interpretation.

It seems to us quite probable that an injury sufficient to produce intracranial hemorrhage may be sufficient to cause alteration of nervous tissue, possibly by pressure of the effused blood; but it is no less likely that intense proliferation of tissue on the inner surface of the dura with the production of more or less hemorrhage disturbs the nutrition of the cortical cells and leads to cellular degeneration. The presence of numerous dilated capillaries and of many red blood cells in the cortex in our case indicate that the nutrition of the cortical cells was imperfect. In addition to this, the brain was very edematous and the edema probably had some effect on the nerve cells. Systematic examination by the best methods of the cerebral cortex in cases of internal hemorrhagic pachymeningitis might frequently reveal cellular changes. Such degenerative changes occur in cases of general paralysis of the insane, chronic alcoholism, etc., and these are conditions in which internal hemorrhagic pachymeningitis is also found.

The nerve cells in our case throughout the central nervous system appear as small distended bladders. The Marchi method applied to the spinal cord shows that the nerve cells here are entirely deprived of any pigment that stains black by osmic acid. The chromatolysis therefore has not given place to pigmentation. Hirsch[15] has described a similar condition as one of toxemia in his case of amaurotic family idiocy, but the disease in our case could not be classed under amaurotic family idiocy. The child was not blind, and microscopical examination shows that the optic nerves were normal. The symptoms were not those of this disease.

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[15] Hirsch. The Journal of Nervous and Mental Disease, 1898.

It has occurred to us that the edema of the cortex might explain the vesicular appearance of the nerve cells, but the cases of uremia reported in literature do not justify this view.

Ewing[16] cites the papers of Acquisto and Pusateri, Sacerdotti and Ottolenghi and Donetti. These writers examined the nerve cells in cases of experimental uremia in animals, but cellular changes such as described by us were not seen by these investigators. Ewing reports six cases of uremia in man, and in some of these the brain and pia were edematous. No cellular changes comparable with those in our case were seen by Ewing. He states also that the effects of pial edema could not be distinctly traced in the cortical cells. Ewing gives no drawings resembling the cells in our case.

It is not impossible that the cachexia was the cause of the cellular changes. Müller and Manecatide[17] have examined the nerve cells of children dying within the first year of life from intestinal diseases, with and without fever. They found cellular changes in all their cases, consisting chiefly of chromatolysis and tumefaction. When the chromophilic elements were destroyed a net of fine fibrils was seen within the cell body. These changes they state resemble those of intoxication and infection, but are not characteristic; neither are the changes of intoxication and infection characteristic, according to Babes and others.

Such a condition of intense tumefaction and chromatolysis might well be supposed to lead to functional disturbance, and it may have stood in causal relation to the feeble mentality, but it seems to have had little influence over motion. Although the motor cells of cortex, medulla oblongata and cord were as much altered as the cells of any other part of the central nervous system, no marked paralysis of motion was noticed. This shows that chromatolysis is not necessarily an index of functional disturbance. Recent investigations lead us to look to the neurofibrils as a very essential portion of the nerve cells, and these have as yet received little attention in studies of pathological processes. The importance of chromatolysis is at present a subject of debate. Nissl[18] himself now acknowledges that chronic poisoning does not produce characteristic changes in the nerve cells, although subacute poisoning with maximal doses gives typical changes from each poison.

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[16] Ewing, Archives of Neurology and Psychopathology, Vol. 1, No. 3, p. 263.

[17] Müller and Manecatide, Zeitschrift für klin. Med., Vol. 36, Nos. 1 and 2, p. 1.

A number of writers have spoken of the absence of functional disturbance, although chromatolysis existed. The experiments of Goldscheider and Flatau[19] are confirmatory of this teaching. Rothmann has recently published some experiments in compressing the abdominal aorta of dogs. In one instance the ganglion cells were much swollen and deprived of chromophilic elements, and yet the function of the posterior limbs shortly before death was almost normal.

We must refer also to Rothmann's[20] statements regarding the reticulum observed by him within the nerve cells. This seems to have been very similar to the network observed by us. Rothmann said the network appeared about ten hours after the compression of the abdominal aorta, and as the Nissl corpuscles disappeared. He thought it was not improbable that this was a previously existing portion of the unstained substance and that it stained in the cells undergoing degeneration, although he did not exclude the possibility of a pathological origin. He refers also to the difference of opinion regarding the normal structure of the cell. Bütschli, Held and Auerbach support the reticular theory of the "neurocytospongium," while Apáthy, Bethe, and Nissl believe that there is a fibrillary character of the cell substance.

Marinesco[21] has reviewed the opinions of various investigators on the structure of the nerve cell, and he states that a network (réseau) exists within the nerve cell. The picture which he gives as figure 1 presents a network very like that seen by us.

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[18] Nissl, Centralblatt für Nerven. u. Psych., Beiheft, 1898, p. 654 and Münch. med. Wochenschrift, No. 31, 1898.

[19] Goldscheider and Flatau, Fortschritte der Medicin, 1897.

[20] Rothmann, Neurologisches Centralblatt, No. 2, 1899, p. 68.

[21] Marinesco, Archiv für Anatomie und Physiologie, Phys. Abtheil, 1899.

It is not our intention to give a thorough review of the literature on the subject of internal hemorrhagic pachymeningitis, as it is exceedingly voluminous. The inflammatory theory seems to have been the older and Calmiel (1826) and Bayle (1826) are mentioned among the early supporters of this view; whereas Andral (1836) and Abercromby (1845) upheld the hemorrhagic theory. The latter theory seems to have found the most support until Virchow (1857) threw the weight of his influence in favor of the inflammatory origin. As Robertson[22] says, a war has been waged by the supporters of these two theories for nearly seventy years, in which upward of ninety writers, mostly French, have taken part. The pendulum has begun to swing a little away from the direction given it by the writings of Virchow; and Laborde, Sperling, Huguenin, Wiglesworth, Dercum, Hoyt, and others have expressed themselves in favor of the hemorrhagic view.

The theory which Wiglesworth[23] has held might be applied to our case. Wiglesworth states that the affection is associated in overwhelming proportion with brain degeneration and atrophy, and that the main cause is the loss of support experienced by the meningeal vessels in the degenerating and wasting brain, assisted by the localized or general congestions of the meninges, so liable to occur in all forms of insanity (p. 434).

Osler,[24] however, believes that something more than atrophy of the brain is needed, or we should find the false membrane more commonly in cases of cerebral wasting.

We hardly feel inclined to enter into a discussion of the views held by Robertson (l. c.), viz., that the all-important element in the production of a subdural membrane is sudden lowering of the intracranial pressure, and that the effect of this is analogous to a dry-cupping of the dura mater. Theories in regard to the formation of internal hemorrhagic pachymeningitis are numerous.

We have spoken of the presence of numerous round, oval and elongated nuclei within the new membrane, but we have not been able to determine the origin of these cells. It is possible, even probable, that they come in part from the white blood cells. In our experiments we found many leucocytes within the forming membrane. The view that spindle cells may develop from the white blood corpuscles has been held by many writers; it was held by Huguenin[25] many years ago, and Wiglesworth,[26] Hoyt (l. c.), and others more recently have supported the same teaching.

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[22] Robertson, The Journal of Mental Science, Vol. XXXIX, p. 203

[23] Wiglesworth, Brain, 1892, Vol. XV, p. 431.

[24] Osler, Journal of Nervous and Mental Disease, 1888, p. 608.

Sperling [27] found that eight days after the injection of fresh coagulable rabbit's blood between the arachnoid and the dura of the rabbit's brain, the clot began to show signs of organization. After two or three weeks the formation of a membrane was complete and in all cases he was able to observe newly formed vessels at this period. This new membrane is said by him to have corresponded in all points with that found in hemorrhagic pachymeningitis.

We have repeated the experiments of Sperling. The animals chosen were dogs and cats. Subdural hemorrhage was produced and the animals were killed at different periods following the experiment; one at three hours, one at six hours, one between six and twelve hours, one at five days, one at seven days, and one at five weeks. In other animals the under surface of the dura was painted with hydrochloric acid and the animals were permitted to live for different periods.

The technique of producing the subdural hemorrhage was as follows: The animal was etherized. A small piece of bone was removed from the left side of the skull, a needle was introduced through the dura and through the superior longitudinal sinus, and hemorrhage was produced in the subdural space on each side of the convexity of the brain. The sections were cut only from the side where the skull remained intact. In the experiments with the acid the same procedure was followed, although the dura of only one side of the brain was painted. Animals in which suppuration occurred were rejected, and only those in which no evidence of a purulent process was found were considered as suitable for our purpose.

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[25] Huguenin, Ziemssen's Handbuch, Vol. XI, part 1, 1876.

[26] Wiglesworth, The Journal of Mental Science, Vol. XXXIII, 1887-1888.

[27] Sperling, Centralblatt für Deutsch. Med., 1871.

A large accumulation of red blood corpuscles was found beneath the dura of a cat killed three hours after the production of a subdural hemorrhage, but no distinct evidences of organization were seen.

A number of polynuclear cells were found in a subdural hemorrhage after six hours.

The evidences of the formation of a new membrane were quite distinct after five days in a subdural hemorrhage in a dog's brain. Many cells with round or oval nuclei, staining deeply with hemalum, were found in the portion of the clot immediately joining the under surface of the dura. These were evidently the nuclei of fibroblasts, although they were much thicker, shorter and more oval than the nuclei of the dura. Similar cells were found in the midst of the clot. In the portion of the clot nearest the pia a mass of amorphous matter, probably fibrin, was found, containing many of these deeply stained nuclei, much smaller and more irregularly formed than those nearest the dura. It seems, therefore, that the organization of the clot begins in the portion nearest the dura, and in the preparations from our case of pachymeningitis in a child, described in this paper, we noticed that the portion of the membrane next to the dura presented a more advanced degree of development.

The new membrane caused by a subdural clot in a dog was very distinctly formed after five weeks. The red blood corpuscles in the clot showed alteration of structure, and beneath these in the portion of the clot nearest the pia a new membrane was found rich in cells. The nuclei of most of these cells were elongated; some were oval or irregularly formed. In one field a distinct capillary could be seen in the new membrane; its endothelial cells were very visible and it contained red blood corpuscles. The great quantity of nuclei in this new membrane were indicative of a very active process.

The sections of the subdural clot from animals permitted to live a few weeks after the formation of a subdural hemorrhage furnish indisputable evidence that a subdural hemorrhage may be *one* of the causes of the formation of a subdural membrane.

An unusual number of cells with elongated nuclei were found on the under surface of a cat's dura on which hydrochloric acid had been used and the animal permitted to live three weeks, but there was no distinct evidence of a newly formed membrane. Even after seven weeks the evidences of a newly formed membrane were not more distinct than in the specimen removed after three weeks. We would conclude from this, that if irritation is capable of producing a false membrane, it must be an irritation that is constant.

The experiments of Goodall[28] seem also to indicate that experimental irritation of the cortex does not produce a false membrane of the dura. He applied sp. vin. gallic. or diluted cantharidin to the cerebral cortex of rabbits through an incision of the dura, and in five animals no inflammatory exudate or naked-eye evidence of inflammation was found; in a sixth a *hemorrhage* was produced in some way and a pseudomembrane formed.

DISCUSSION.

Dr. E. D. Fisher thought that the early idea that hemorrhagic pachymeningitis was rare was due, as Osler said, to observing the wrong class of cases. In any almshouse this condition would be found very prevalent. Some years ago the question came up in the New York Pathological Society, and he took occasion at that time to show a number of cases of this disease. He did not quite understand the etiology of the case reported by Drs. Spiller and McCarthy, and asked for further information.

Dr. D. J. McCarthy replied that the etiology of the case was rather obscure, there being evidence both of rachitis and syphilis. Diarrhea existed for a short time before death, but that probably had no causative relation to the membrane itself; the etiological factors were syphilis and rachitis.

Dr. McCarthy stated that the statistics show that 2.7 per cent. of the entire number of cases of pachymeningitis occur during the first year of life.

Dr. W. G. Spiller said that cases of internal hemorrhagic pachymeningitis are not very uncommon in the insane, as Dr. Fisher had stated, but they occur much less frequently in childhood, and he believed that no case in a child so old as nine years had been published. In the case just reported, the flat bridge of the nose and the Hutchinson teeth were indicative of syphilis; and the so-called pigeon-breast was a sign of rachitis.

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[28] Goodall, The Journal of Mental Science, Vol. XXXVIII, p. 397.